

Investigation vignette

A 57 Year old Woman Admitted to the Emergency Department with Hyponatraemia and Hypoglycaemia

CASE REPORT

A 57 year old lady was admitted to the emergency department with a depressed level of consciousness. Through an interpreter, her family reported that she had become increasingly drowsy over the last 2 days. Three months previously she had been discharged from hospital following an episode of gastroenteritis.

A neurological examination revealed a Glasgow coma score (GCS) of 8 (M5 V2 E1), with no localising signs. Her blood pressure was 110/70 mmHg, pulse

110 beats per min and her temperature was 36°C. The physical examination was otherwise unremarkable.

A semiquantitative examination for blood glucose (BM-test®) was recorded as low and so an intravenous infusion of dextrose was administered. Following this her GCS increased to 11 (M5 V2 E4).

A biochemical profile performed in the emergency department (Figure 1) led to the diagnosis.

Name	Age	Sex	Time of Collection Analysis		Date
Mrs. B. T.	57	F	1250	1300	11.8.98

Sodium	105	mmol/L	(135 - 146)
Potassium	3.6	mmol/L	(3.5 - 5.0)
Chloride	79	mmol/L	(95 - 110)
Glucose	1.3	mmol/L	(3.0 - 6.0)
Urea	3.2	mmol/L	(3.2 - 7.7)
Creatinine	0.10	mmol/L	(0.05 - 0.12)
Albumin	34	g/L	(35 - 47)
Total bilirubin	20	µmol/L	(2 - 20)
ALT	22	U/L	(0 - 45)
ALP	40	U/L	(25 - 120)
CK	5640	U/L	(30 - 180)

Figure 1. Plasma electrolytes, glucose and liver function tests taken from a blood specimen on admission to the emergency department

Diagnosis: Panhypopituitarism with secondary hypothyroidism and adrenocortical insufficiency

The combination of an elevated serum creatine kinase (CK), hyponatraemia and hypoglycaemia suggested hypothyroidism and adrenocortical insufficiency (e.g. anterior pituitary insufficiency). Further investigation revealed a plasma free T₄ 0.0 pmol/L (normal range 10 - 24 pmol/L), TSH 1.8 mU/L (normal range 0.4 - 4.0 mU/L), cortisol 100 nmol/L (peak diurnal level 200 -700 nmol/L) prolactin 19 mU/L (normal range 10 - 650 mU/L), FSH 1 IU/L (normal range 25 -120 IU/L) and LH < 1 IU/L (normal range 16 - 80 IU/L).

The cause of the elevated CK (predominantly the isoenzyme form CK-MM) with hypothyroidism is uncertain but may be related to cellular leakage resulting from increased cell membrane permeability.¹ Hypothyroidism can also be associated with elevations in other serum enzymes such as aspartate aminotransferase and lactate dehydrogenase,² as well as elevations in serum magnesium and cholesterol, although these were not measured in this patient.

The casual relationship between hypothyroidism and hyponatraemia has been recently challenged, as one report found no relationship between the serum TSH and sodium levels.³ Nevertheless, hyponatraemia and primary hypothyroidism is a well-recognised phenomenon,⁴ with the hyponatraemia resulting from water retention, due to both arginine vasopressin (AVP)⁵ and non AVP mechanisms.⁴ However, hyponatraemia associated with anterior pituitary disease may be largely due to ACTH deficiency, as it may be corrected by glucocorticoid rather than thyroid replacement.⁴

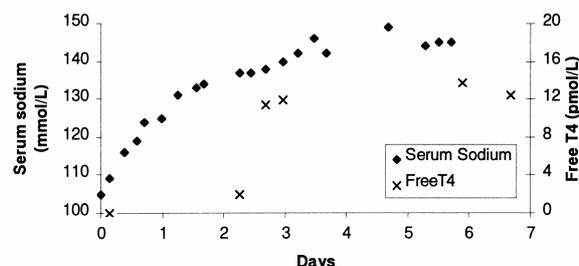
Due to the loss of counter-regulatory hormones, hypoglycaemia is more commonly seen in panhypopituitarism (secondary adrenocortical insufficiency) than in Addison's disease.⁶ However, apart from mild hyponatraemia, fluid and electrolyte abnormalities are not characteristic of secondary adrenocortical insufficiency,⁶ because aldosterone secretion is relatively independent of pituitary function.

A CT head scan was performed in this patient which revealed changes suggestive of the 'empty sella syndrome'.

The patient was admitted to the intensive care unit and treated with hydrocortisone (100mg 8-hourly) and

thyroxine (50µg daily). There was a spontaneous diuresis and the serum sodium returned to the normal range within 48 hours. Three days after admission the free T₄ was also within the normal range (Figure 2). The serum CK was normal by day six of the admission.

Figure 2. Sodium and T4 changes after admission



Despite this relatively rapid metabolic improvement the patient remained drowsy, however over the next two weeks her conscious state improved. She was discharged to the medical ward with a mild cognitive impairment from which she subsequently fully recovered.

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